# Diet Induced Thermogenesis

Diet-Induced Thermogenesis (DIT) is the production of heat that occurs after eating - which contributes to the body's resting metabolic rate. DIT is also called the thermic effect of food. It activates sympathetic nervous system activity and increases Resting Metabolic Rate. Overeating alone can increase caloric expenditure. Most people though consume far more calories than they burn even with the increased metabolic response. Although DIT is sometimes questioned in humans most experts agree that it accounts for 3-10% of daily energy expenditure.

The mechanism for DIT has two components which contribute to energy expenditure. Immediately after eating and for a period of several hours the body expends more energy to support the ingestion, digestion, and absorption of nutrients as well as transport of blood. This is the mechanical element of diet-induced thermogenesis. In addition, the body produces heat in brown adipose tissue (BAT) due to increased sympathetic nervous system activity. This process, though, is not completely understood.

BAT is a unique form of fat that is found in varying amounts throughout the mammalian genus. Bears, rodents and other hibernating animals that live in cold weather have an abundance of this specialized fat. Humans have small deposits throughout the body. BAT cells are smaller than normal storage cells, contain less fat, and are laden with mitochondria. These mitochondria hold a specialized uncoupling protein that uncouples the hydrogen atoms from the respiratory chain, producing heat but no ATP for work. The mechanism is based on a sympathetic response to cold. Norepinephrine is released from nerve endings in the BAT. In turn the uncoupling protein uncouples oxidative phosphorylation so ATP is not generated but heat is.

Brown adipose tissue is stimulated through two primary mechanisms, cold environment and food consumption. The cells respond to the changing temperature, which stimulate the production of heat. Unlike storage cells these adipocytes experience hyperplasia in response to the stimulus. This means they actually increase in number, as well as size.

Food consumption can activate this protein in brown adipose tissue in a similar manner. The magnitude of the DIT may depend on the number of calories consumed as well as the type of nutrient ingested. When a study on non-obese young males was performed the researchers found that calories ingested in two meals had a lower thermic response than the same number of calories eaten across four meals. The smaller meals required an additional caloric expenditure of over sixty calories above that expended for the two larger meals of the same caloric content. In addition, they found that carbohydrates have a greater thermic effect than fats and proteins have a greater effect than the carbohydrates. It is thought that the conversion of glucose to glycogen and the synthesis of body proteins that occurs after protein ingestion cause the increased metabolic demand.

Some foods contain products that stimulate metabolism without calories. Foods and beverages containing caffeine can elevate the body's metabolism. This stimulant is found in chocolate, soda, and coffee. One cup of American coffee can increase metabolism by 3-4% for a short period of time. Likewise, spicy foods can have a similar effect. Capsaicin found in hot peppers has a pronounced thermic effect. Both caffeine and capsaicin stimulate the sympathetic nervous system to cause the enhanced metabolic activity.

Thermic activity can partially explain individuals that have the ability to consume

high amounts of food without gaining fat. Human infants are believed to maintain more BAT to aid in the maintenance of body. Again there is some question as to the amount and actual role BAT plays in adult humans but there is evidence that other uncoupling proteins exist in other tissues of the body.

UCP-1 is the uncoupling protein found in the BAT. Recently other uncoupling proteins have been found in fat storage cells, the brain and muscles. These proteins are called UCP-2 and UPC-3, respectively. This finding helps to explain how, even with minimal amounts of brown fat, people may be able to accelerate their metabolism to compensate for overeating. In obese individuals it is theorized that these uncoupling proteins are defective and fail to enhance metabolism with the consumption of food.

When laboratory rats are overfed they gain less weight than is expected based on the level of positive caloric balance. Scientists attribute this to activation of the sympathetic nervous system in BAT, causing an accelerated metabolism. The rodents have shown an increase in BAT in response to the overeating. Interestingly, some rats fail to increase BAT with the food consumption and become obese. Investigations using humans have been less definitive than the studies using animals, which may explain the uncoupling proteins found in other areas of the human body.

The problem with analyzing humans in the same manner we analyze animals is obvious. But, a study done in the early seventies gave scientists a more direct look at this phenomenon. The scientists used human cadavers ranging in age up to 80. The evidence suggests that humans have a similar response to the thermogenesis found in animals. The study showed that up until age 10 brown adipose tissue is widely distributed throughout the body. By the age of thirty though, most brown adipose tissue disappears from the body. The brown fat that remains well into old age mainly surrounds central organs, and is found in the chest cavity and neck.

In most studies performed on living humans the evidence fails to show high statistical significance. This is often due to the study methodology and procedures used. Many times the humans in the studies were not adequately measured, unequal food consumption was common, factors such as exercise, smoking and nutritional status all varied. These factors would all lead to inconclusive data collection. Almost all studies though, showed varying amounts of diet induced thermogenesis. With most people reaching peak DIT within four hours after eating and lean individuals peaking even earlier.

Weight gain was originally thought to be only a function of calories. This though is not the case. If it were we could adjust calories and easily predict an outcome. A study done on prisoners in a Vermont correctional facility did just that. Scientists modified the diets of the volunteers from the prison population based on their relative metabolisms. They found an initial increase in weight gain to be 21% of the expected based on the calories consumed. They were further astonished by the fact that as the trial went on the men had more difficulty gaining additional weight. Some actually lost weight. They found that to increase body weight to a greater degree they had to increase caloric intake well over 7000 kcal.

The study found a great variability in each individual's ability to gain weight. Although the DIT was not measured the scientists concluded that the thermic affect of the food was a main contributor to this response. There are several theories as to the reasons for this phenomenon. The popular speculation being that human ancestors developed this defense against obesity when food consumption was high to maintain adequate nutrition from foods holding only trace amounts of essential nutrients.

Although the link between obesity and a defective thermic response have not been proven there is quite a bit of evidence that

points to this possibility. If there is a high variance in DIT in all humans due to the uncoupling proteins found throughout the body many of the variances in weight gain could be explained. This may also give more credibility to theories such as “Set-point” which uses predisposed genetic factors as the explanation for weight gain and difficulties with weight loss. Never-the-less, exercise and a calorie controlled healthy diet, low in fat and high in nutrient rich carbohydrates can help prevent the occurrence of obesity in most people.